group individuals for the prevalence of HIV infection through a network of surveillance centres.[2] As a part of such a surveillance programme 7050 high risk group individuals, namely 4957 STD clinic patients (3594 males and 1363 females, 1195 blood donors (1144 males and 51 females), 54 female prostitutes, 820 antenatal cases and 24 contacts of HIV infected cases, were screened to find the prevalence of HIV infection at Tirupati, a renowned Hindu pilgrim town in South India which is about 150 km from Madras and Vellore where a high prevalence of HIV infection has been reported.[2]

Serum samples were collected from these individuals and tested for the presence of HIV antibodies by ELISA using Wellcozyme brand kits. Repeatedly ELISA positive sera were subjected to a confirmatory Western Blot Test.

Out of 7050 samples screened, 50 were seropositive (0·71%). Among 3594 male STD patients, 31 (0·86%) were seropositive. However, among 1363 female STD patients who were either contacts or wives of male STD patients no HIV seropositivity was detected. In contrast to this, a high prevalence of HIV infection was found among 14 (25·92%) of 54 female prostitutes tested, which reinforces the belief that the risk of acquiring HIV infection is greater in persons having a large number of sexual partners.[3] Out of 1144 male blood donors three (0·26%) were seropositive and among 51 female blood donors seropositivity was not noticed. Among 820 antenatal cases tested 2 (0·24%) were positive. None of the regular contacts or spouses of 50 HIV seropositive individuals were seropositive, thereby indicating seropositivity need not be associated with infectivity.[4] The incidence of HIV seropositivity among high risk group individuals screened in different parts of India varies greatly from 0·03% to 0·54%.[5] Factors like life style, nutritional status, nature of infectious co-factors and differences in the genetic make up may be responsible for such variations.[6]

Globally now there are several ongoing studies to define the spectrum of seropositive cases both in rural and urban areas. HIV infection and AIDS cannot be controlled anywhere if efforts are not made everywhere. There is hope that the future will permit control and cure if not eradication of HIV infection.

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Endemic syphilis in Bulgaria

Between 1958 and 1962 an eradication campaign against endemic syphilis, using 10 to 15 days course of injectable penicillin, was carried out in Bulgaria. The subjects were predominantly of Moslem Turkish origin. In all 608,816 persons were examined and serological tests for syphilis performed. Of these persons 6,290 (1·0%) were found to have endemic syphilis.

To see if the previous campaign had been successful, a further study was made between October 1989 and March 1990 on 27,435 Bulgarian Moslems of the same background as the group of 1958–1962. A total of 23 persons were found to have positive serological results of whom four were due to primary, secondary or early latent syphilis, leaving 19 (0·07%) of 27,435 who were thought to have endemic syphilis.

The frequency of endemic syphilis had diminished to one fifteenth of that in 1958–1962. The mean age of those with endemic syphilis was 56·3 years, 11 were men, eight were women. All were country dwellers with limited education. It is possible that some persons with endemic syphilis have died since the previous survey or emigrated to Turkey.

The conclusion is that endemic syphilis is more or less eradicated in Bulgaria. There may be factors in its disappearance other than medical intervention, such as a general increase in living standards.

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Incidence of *Neisseria gonorrhoeae* and *Chlamydia trachomatis* infections in Strasbourg over 8 years

As described in many western countries, the incidence of sexually transmitted diseases (STD) has been decreasing since 1980 and this phenomenon is assumed to be related chiefly to the emergence of HIV infections.[1]

The incidence of infections due to *Neisseria gonorrhoeae* and *C. trachomatis* at the STD Dispensary (located within the Dermatology Department) of Strasbourg over 8 years (from 1983 to 1990), was collected from the annual activity reports. For diagnosis, samples were obtained from the anterior urethra in male and from cervix in women. Since 1983, diagnosis of chlamydial infections have been done using cycloheximide treated McCoy cells. However, since 1986, an enzyme immunosorbant assay (Chlamydiazyme, Abbott Laboratories, Chicago) was used concurrently: from 1986 to 1989, 3046 cell cultures (355 positive, 11·7%) and 3128 Chlamydiazyme (232 positive, 7·4%) assays were performed.

The results from these two methods are considered separately. *N gonorrhoeae* was isolated as previously described.[2] Isolation of *N. gonorrhoeae* was two times more frequent in men than in women. For *C. trachomatis* infections the difference between sexes was less important. From 1983 to 1989 (table), the incidence of gonococcal infections diagnosed in the dispensary has decreased regularly. This phenomenon was accompanied by a diminishing attendance at the consultation. The incidence of *C. trachomatis* infections varied but overall remained in the same range. Since 1986, diagnosis of chlamydial infection was performed more systematically: an apparent drop of the positivity rate can be noticed for this year; however, the same number of infections was diagnosed in 1985 (187) and 1986 (183).

Cell cultures gave a higher positivity rate when compared with Chlamydiazyme in men but this rate does not vary significantly in women. If a lower sensitivity of Chlamydiazyme may be invoked, there is another reason for such a difference: cell culture is performed for patients who can afford its cost; for the others, Chlamydiazyme is used as the administration of the Department of the Bas-Rhin (which rules the Dispensary) pays for this test which is far less expensive.

When analysing the table, a moderate diminution of the incidence of chlamydial infection can be noted only in women, when considering cell culture since 1983. This contrasts with the reports in English women, of a low prevalence of *C. trachomatis* infections in one STD dispensary[3] or a simultaneous decrease of *C. trachomatis* and *N. gonorrhoeae* prevalence in the other.[4] In Strasbourg, the ratio chlamydial/gonococcal infections in men has increased from about 1 in 1984 up to 8 in 1990 and in women from 2 up to 7.

The differences between these reports might be due to the populations screened. However, it is also possible that *N. gonorrhoeae* and *C. trachomatis*

Table Incidence of Chlamydia trachomatis and Neisseria gonorrhoeae infections at the Dispencary over 7 years

(a) Chlamydia trachomatis

	Men				*Women*			
	Culture		*Chlamydiazyme*		*Culture*		*Chlamydiazyme*	
	Number	*% positive*	*Number*	*% positive*	*Number*	*% positive*	*Number*	*% positive*
1983	225	18·9	–		230	10·9	–	
1984	288	20·1	–		294	14·6	–	
1985	538	19·5	–		454	18·1	–	
1986	446	15·7	370	8·4	355	10·4	495	9·1
1987	661	11·8	271	5·3	495	8·6	355	7·0
1988	384	7·3	376	4·6	226	7·9	321	7·7
1989	169	14·7	291	5·5	90	4·4	244	9·0
1990	133	17·3	194	7·2	74	6·7	231	9·5

(b) Neisseria gonorrhoeae

	Men		*Women*	
	Culture number	*% positive*	*Culture number*	*% positive*
1983	1343	16·2	1230	7·9
1984	1393	14·9	1238	7·8
1985	1387	11·6	1010	3·9
1986	1315	7·2	1112	3·9
1987	1122	3·3	1057	1·7
1988	1171	2·5	749	0·5
1989	857	2·0	582	0·5
1990	642	2·2	520	1·0

possess different epidemiological characteristics.

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Female paraurethral ducts and glands as the sites of agents of sexually transmitted diseases

Female paraurethral (Skene's) ducts and paraurethral glands (PUDG) and the male prostate are morphologically and functionally identical organs, as has been proved by excellent studies of Zaviačič *et al.*[1-6] Thus, PUDG and the male prostate could both be affected with inflammatory changes caused by sexually transmitted agents. We report two cases of female "prostatitis" of trichomonad aetiology.

The first case was a 26 year old waitress, multipara $\times 2$, married. Two years previously she had been treated, together with her husband, for trichomonas vaginitis with metronidazole 2·0 g orally in a single dose. Since then she reported repeated swelling of the vaginal wall in the location of the PUDG and urethra. After pressure on this area, pus was expressed from the urethra. The condition was accompanied by dysuric symptoms. Lactobacillus species and coagulase negative staphylococci were isolated from vaginal culture using Thayer Martin medium, Sabouraud glucose agar, MacConkey's agar and trichomonas medium (Oxoid). After opening of the urethral meatus, to a depth of 4 mm from the margin of the external orifice of the female urethra, the orifices of the PUDG were examined colposcopically, enlarged 12·5 times and appeared as two symmetrically set pointed orifices with inflammed margins and fibrin residue. After pressure on the vaginal wall against the urethra in the direction from the depth of the urethra to the external orifice of the urethra, 5 ml of pus from PUDG were expressed. From the pus culture using the same culture media, *Streptococcus agalactiae* was isolated. The patient was treated for 10 days with ampicillin 3·0 g daily in accordance with the sensitivity. The process recurred and a postinflammatory obliteration of the PUDG lumina developed. Owing to retention of the contents a painful swelling the size of a hen's egg occurred in the proximal third of the vagina and PUDG orifice. The white blood cell count was $12{\cdot}1 \times 10^9/l$ and erythrocyte sedimentation rate was 50 mm in one hour. Transvaginal surgical extirpation of the tumour was necessary.

After incising the abscess cavity, *Trichomonas vaginalis* and *Streptococcus agalactiae* were isolated from pus by culture. Histological examination proved that the abscess cavity was a dilated, inflamed, paraurethral gland with postinflammatory obliteration of the duct. Thanks to a 10 day treatment with ampicillin 2·0 g daily and metronidazole 750 mg daily the inflammation has been successfully controlled.

The second case was a 43 year old female worker, with three children, divorced. She stated that she last had sexual intercourse a year previously when she was treated for trichomonas vaginitis by metronidazole 2·0 g orally in a single dose. The PUDG inflammation, similar to that in the first case, was clinically manifested by painful swelling of the proximal third of the vagina in the location of the PUDG lumen, with dysuric problems and subfebrile states, with no changes in leukocyte count or erythrocyte sedimentation rate. Lactobacillus sp., *Eubacterium lentum* and coagulase negative staphylococci were isolated from vaginal culture. Under colposcopic control, enlarged 12·5 times, probing of the PUDG duct broke the adhesions, and after pulling out the probe from a depth of about 0·5 cm, approximately 4 ml of pus spontaneously flowed out through the orifice of the PUDG. From the pus culture *Trichomonas vaginalis* was isolated. After a 10 day treatment with ampicillin 2·0 g daily and metronidazole 750 g daily the inflammation resolved without surgical intervention.

These cases of PUDG inflammation of trichomonad aetiology indicate that the "female prostate" can be one of the sites of STD. Furthermore, this localisation can also be the cause of relapsing STD.

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